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CLAIMS

1. Use of a TNF-α inhibitor selected from the group consisting of:
- metallo proteinase inhibitors excluding methylprenisolone,
- tetracyclines including chemically modified tetracyclines,
- quinolones,
- corticosteroids,
- thalidomide,
- lazaroides,
- pentoxyphyllines,
- hydroxamic acid derivatives,
- carbocyclic acids,
- napthopyrans,
- soluble cytokine receptors,
- monoclonal antibodies towards TNF-α,
- amrinone,
- pimobendan,
- vesnarinone,
- phosphodiesterase III inhibitors,
- lactoferrin and lactoferrin derived analogous, and
- melatonin

in the form of the base or its addition salt, in the preparation of a pharmaceutical composition for the treatment of spinal disorders as nerve root injury caused by the liberation of TNF-α and compounds trigged by the liberation of or presence of TNF-α by inhibiting spinal disk TNF-α.

2. Use of a TNF-α inhibitor in the form of a soluble cytokine receptor in the preparation of a pharmaceutical composition for the treatment of spinal disorders as nerve root injury caused by the liberation of TNF-α and compounds trigged by the liberation of or presence of TNF-α by inhibiting spinal disk TNF-α.

3. Use according to claim 1 or 2, wherein the TNF-α inhibitor is the soluble cytokine receptor etanercept.

4. Use of a TNF-α inhibitor in the form of a monoclonal antibody towards TNF-α in the preparation of a pharmaceutical composition for the treatment of spinal disorders as nerve root injury caused by the liberation of TNF-α and compounds trigged by the liberation of or presence of TNF-α by inhibiting spinal disk TNF-α.

5. Use according to claim 1 or 4, wherein the TNF-α inhibitor is the monoclonal antibody infliximab.

6. Use according to claim 1, wherein the TNF-α inhibitor is selected from the group consisting of tetracycline, doxycycline, lymecycline, oxytetracycline, minocycline, and chemically modified tetracyclines dedimethylaminotetracycline, in the form of bases or addition salts.

7. Use according to claim 6, wherein the TNF-α inhibitor is doxycycline.

8. Use according to claim 1, wherein the TNF-α inhibitor is selected from hydroxamic acid compounds, carbocyclic acids and derivatives, thalidomide, lazaroides, pentoxyphylline, napthopyrans, amrinone, pimobendan, vesnarinone, phosphodiesterase III inhibitors, melatonin in the form of bases or addition salts.

9. Use according to claim 1, wherein the TNF-α inhibitor is selected from norfloxacine, ofloxacine, ciprofloxacine, gatifloxacine, pefloxacine, lomefloxacine, and temafloxacine in the form of bases or addition salts.

10. Use according to claim 1, wherein the TNF-α inhibitor is a metallo proteinase inhibitor in the form of base or addition salts.

11. Use of a substance inhibiting a compound trigged by the release of TNF-α, such as interferon-gamma, interleukin-1, and nitrogen oxide (NO), in the form of base or addition salts in the preparation of a pharmaceutical composition for the treatment of spinal disorders as nerve root injury caused by the liberation

of TNF-α and compounds trigged by the liberation of or presence of TNF-α by inhibiting spinal disk TNF-α.

12. Use according to any one of the claims 1-11, wherein said nerve root injury is induced by disk herniation.

13. Use according to any one of the claims 1-11, wherein said nerve root injury is nucleus pulposus-induced.

14. Use according to claim 12 or 13, wherein said nerve root injury is sciatica.

15. A pharmaceutical composition for the treatment of nerve root injury comprising a pharmaceutically effective amount of a soluble cytokine receptor.

16. A pharmaceutical composition according to claim 15, wherein said soluble cytokine receptor is etanercept.

17. A pharmaceutical composition for the treatment of nerve root injury comprising a pharmaceutically effective amount of a monoclonal antibody selective for TNF-α.

18. A pharmaceutical composition according to claim 17, wherein said monoclonal antibody is infliximab.

19. A method for partially blocking nucleus pulposus-induced reduction of nerve conduction velocity, comprising the administration of a blocking-effective amount of a monoclonal antibody selective for TNF-α.

20. A method according to claim 19, wherein said monoclonal antibody is infliximab.

21. A method for the treatment of spinal disorders as nerve root injury caused by the liberation of TNF-α in mammals, including man, comprising the administration of a pharmaceutically effective amount of a TNF-α inhibitor selected from the group consisting of:

- metallo proteinase inhibitors excluding methylprenisolone,
- tetracyclines including chemically modified tetracyclines,
- quinolones,
- corticosteroids,

- thalidomide,
- lazaroides,
- pentoxyphyllines,
- hydroxamic acid derivatives,
- carbocyclic acids,
- napthopyrans,
- soluble cytokine receptors,
- monoclonal antibodies towards TNF-α,
- amrinone,
- pimobendan,
- vesnarinone,
- phosphodiesterase III inhibitors,
- lactoferrin and lactoferrin derived analogous, and
- melatonin

in the form of the base or its addition salt.

22. A method for the treatment of spinal disorders as nerve root injury caused by the liberation of TNF-α in mammals, including man, comprising the administration of a pharmaceutically effective amount of a TNF-α inhibitor in the form of a soluble cytokine receptor.

23. A method according to claim 21 or 22, wherein said TNF-α inhibitor is the soluble cytokine receptor etanercept.

24. A method for the treatment of spinal disorders as nerve root injury caused by the liberation of TNF-α in mammals, including man, comprising the administration of a pharmaceutically effective amount of a TNF-α inhibitor in the form of a monoclonal antibody towards TNF-α.

25. A method according to claim 21 or 24, wherein said TNF-α inhibitor is the monoclonal antibody infliximab.

26. A method according to claim 21, wherein the TNF-α inhibitor is selected from the group consisting of tetracycline, doxycycline, lymecycline, oxytetracycline, minocycline, and chemically modified tetracyclines dedimethylaminotetracycline, in the form of bases or addition salts.

27. A method according to claim 26, wherein the TNF-α inhibitor is doxycycline.

28. A method according to claim 21, wherein the TNF-α inhibitor is selected from hydroxamic acid compounds, carbocyclic acids and derivatives, thalidomide, lazaroides, pentoxyphylline, napthopyrans, amrinone, pimobendan, vesnarinone, phosphodiesterase III inhibitors, melatonin in the form of bases or addition salts.

29. A method according to claim 21, wherein the TNF-α inhibitor is selected from norfloxacine, ofloxacine, ciprofloxacine, gatifloxacine, pefloxacine, lomefloxacine, and temafloxacine in the form of bases or addition salts.

30. A method according to claim 21, wherein the TNF-α inhibitor is a metallo proteinase inhibitor in the form of base or addition salts.

31. A method for the treatment of spinal disorders as nerve root injury caused by the liberation of TNF-α and compounds trigged by the liberation of or presence of TNF-α in mammals, including man, comprising the administration of a pharmaceutically effective amount of a substance inhibiting a compound trigged by the release of TNF-α, such as interferon-gamma, interleukin-1, and nitrogen oxide (NO), in the form of base or addition salts.

32. A method according to claim 21, wherein said nerve root injury is induced by disk herniation.

33. A method according to claim 21, wherein said nerve root injury is nucleus pulposus-induced.

34. A method according to claim 21, wherein said nerve root injury is sciatica.

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